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ORIGINAL ARTICLE

Ocular Manifestations in Psoriasis: The Importance of Ophthalmological Examination

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Abstract: Psoriasis is a chronic, inflammatory, immune-mediated skin disease affecting 1-3% of the adult population. It mainly involves the skin, nails, and joints. However, psoriasis can be associated with several comorbidities, including ocular complications. The most frequently reported pathologies are dry eyes syndrome, blepharitis, and uveitis. Being a systemic inflammatory disease, psoriasis also has the potential to directly affect the retina. It has been reported that ocular manifestations tend to appear much later than skin and joint involvement. Eye complications begin insidiously, and progress asymptomatically. This can lead to a decrease in the quality of life and even permanently compromised visual function if not treated properly. Thus, the present review aimed to present current pathological explanations between psoriasis and ocular manifestation, and to evaluate the prevalence of ocular changes in patients with psoriasis, in order to be diagnosed and managed appropriately.

Keywords: psoriasis, systemic disease, parakeratosis, ocular complications, uveitis, dry eye syndrome, retina, cornea, retina, cataract

INTRODUCTION

Psoriasis is a common chronic, inflammatory, immune-mediated skin disease, affecting both sexes equally, but men usually have more severe forms of the disease than women [1]. Skin manifestations are characterized by the presence of very well-circumscribed, erythematous papules or plaques with a grey or silvery-white, dry scale, over the surface of the skin with different morphology, distribution, and severity. The lesions are often distributed symmetrically on the elbows, knees, lumbosacral area, and scalp [2].

Psoriasis affects 1-3% of the adult population and negatively impacts the quality of life of the patients [2]. While it primarily affects the skin, psoriasis is associated with several comorbidities, such as psoriatic arthritis, Crohn's disease, cardio and cerebrovascular disorders, and ocular diseases, which confirms that psoriasis is a systemic entity [3]. Ophthalmological manifestations in psoriasis have an occurrence rate of 10-12% involving different parts of the eye and presenting various symptoms [4]. Key ocular manifestations associated with psoriasis include uveitis, dry eye, retinal abnormalities, blepharitis, conjunctivitis, keratitis, iridocyclitis, and UV-induced cataracts [3]. In particular, patients with psoriatic arthritis show a higher risk of developing uveitis than psoriasis patients without joint involvement [3]. Moreover, psoriasis treatments may also lead to ocular complications, with a higher prevalence of ocular manifestations in patients treated with systemic treatments such as methotrexate, acitretin, and biologic drugs, and less frequent in cases treated with topical steroids and phototherapy [3]. However, ocular manifestations of psoriasis are often nonspecific or mildly symptomatic and may be often misdiagnosed [3].

PHYSIOPATHOLOGY OF PSORIASIS AND OCULAR COMORBIDITIES

Physiopathology of psoriasis

Psoriasis is a yet incompletely understood condition in which an unspecified stimulus triggers the activation of innate immune defense, with differentiation of proinflammatory M1-type macrophages. These in turn activate the stimulation of innate immune defense chains via IL23, which leads to the emergence of LyTh1 and LyTh17 cell clones with the release of IL17. At the tissue level, keratinocyte hyperproliferation, vascular hyperplasia, as well as the formation of tissue memory Ly Th and the reduction of regulatory Ly Th activity occur. Although there is now a

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wealth of data illustrating the etiopathogenic mechanism in psoriasis, the initial triggering of the immune system is unknown. It is hypothesized that environmental factors, a change in the composition of the skin microbiota, or other types of stressors may be at the origin, but in the absence of concrete data, we can only speculate.

From the present information, however, we are well aware of the close interrelationships between the cutaneous dendritic cell, neutrophils, macrophages, and the acquired immune system, with the triggering of immune relays leading both to the clinical manifestations of the disease and the formation of a local immunologic scar [5].

On exacerbation or onset of psoriasis, activated dendritic cells produce interleukin 12 (IL-12), IL-23, and TNF-alfa. IL-23 activates Th17 cells to produce IL-17, which stimulates keratinocytes to proliferate in an alternative pathway. Sustained inflammation leads to uncontrolled proliferation and abnormal differentiation of keratinocytes [6].

Potential physiopathology of ocular comorbidities

The exact mechanism by which psoriasis affects various parts of the eye is not fully known, but it is known that the skin and eye share a common origin from the ectoderm [4]. Thus, it is hypothesized that the ocular manifestations are closely associated with the cutaneous manifestations through an immune-mediated mechanism [4]. There are a few possible pathological mechanisms that can account for the ocular manifestations in psoriasis patients. The first explanation is the increased inflammatory reaction in psoriasis, which can trigger ocular inflammation [6]. The second is genetic predisposition, HLA B-27, which could lead to autoimmunity in both the skin and the eye [6].

MATERIALS AND METHODS

A structured literature search of titles in PubMed was performed in October 2024. The literature search was performed according to the updated preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines [7]. The keywords used for finding relevant results were: “psoriasis”, “ocular manifestations”, “psoriatic”, “uveitis and psoriasis”, “ophthalmology” and all relevant synonyms, abbreviations, and combinations between these terms. The references of identified articles were manually checked to find potentially relevant studies. All studies, articles, and systematic reviews published before 2014 were excluded. All studies, articles, and systematic reviews concerning ocular side effects of the treatments used in psoriasis were excluded. The bibliography of these articles or reviews was analyzed by the authors and if relevant or complementary articles for this review were found, conducted to their integration in this study regardless of the year of publication. Studies were included for full-text review and qualitative analysis if they reported on well-known ocular manifestations of psoriasis, rare associations between psoriasis and ocular findings, as well as novel approaches of immunological links between psoriasis pathological mechanisms and subsequent clinical ocular affections. The authors acknowledge that this search string potentially may have unknowingly excluded important research papers.

RESULTS AND DISCUSSIONS

Being a chronic inflammatory disorder, psoriasis could manifest with various extra-cutaneous manifestations of which eye involvement is important. The ocular structures that could be directly or indirectly touched by the inflammatory imbalance induced by psoriasis are conjunctiva, Meibomian glands, eyelids, cornea, sclera, uvea, and retina [8]. From this enumeration, it is obvious that psoriasis could affect every ocular structure. Each of these ocular structures has a clinical impact on the patient and creates a broad spectrum of manifestations including relatively mild ocular disorders such as conjunctival hyperemia and severe ocular disorders such as corneal melting or retinal affections which could lead to vision loss [9].

Discussing ocular changes induced by a parakeratosis disease, we will structure the results depending on the presence of epithelium in the affected ocular structures. We classify the ocular structures into two categories: 1. External ocular structures- epithelium-containing structures, which could be exposed to the parakeratosis effect of psoriasis (conjunctiva, cornea, meibomian glands) and 2. Internal ocular structures -without epithelium exposed to parakeratosis effect (uvea, retina). This classification can define better the proportions of inflammatory status solely which subsequently affects the ocular structures and its combination with the epithelial damage.

External ocular structures

Conjunctival manifestations

The most frequent manifestation of psoriasis in conjunctiva is conjunctivitis [2]. Symptoms of conjunctivitis include conjunctival redness, tearing or thick yellow discharge, ocular pain, or the feeling of grittiness or foreign bodies in the eyes. The signs of conjunctivitis can include conjunctival hyperemia, demarcated, yellowish-red plaques on the palpebral conjunctiva, or areas of xerotic appearance on the bulbar conjunctiva [2]. After reviewing the literature, it was not possible to find a study that attests to the exact mechanism of the occurrence of conjunctivitis in psoriasis. Therefore, it is not known whether conjunctivitis appears as a result of the specific epithelial damage in psoriasis or as a result of systemic inflammation. Even though psoriasis commonly can cause conjunctivitis, it is important to remember that this condition is more commonly due to allergies, bacterial infection, or viral infection [10]. Regardless of the pathological mechanism of conjunctivitis in psoriasis, left untreated, conjunctivitis complicates itself with xerosis, symblepharon, or trichiasis which could lead to further complications involving the cornea [9].

Posterior Blepharitis

The cause of posterior blepharitis is the meibomian gland dysfunction (MGD). Meibomian glands (MG) are sebaceous glands located in the eyelids. They are responsible for secreting the lipid component of the eye tear film which plays an important role on the ocular surface by preventing the evaporation of tears [11]. Meibomian gland dysfunction is a chronic disease characterized by terminal duct obstruction and/or quantitative-qualitative changes in secretions [12]. The mechanism that links these two conditions is not known, but it was proposed that an increased epithelial turnover leads to high volumes of cell production and subsequent shedding that may ultimately lead to a mechanical block of the meibomian duct [2,13].

Regardless of the exact mechanism that links psoriasis and blepharitis, the epidemiological data suggest a prevalence rate of MGD in psoriasis patients even as high as 59.2% [14]. Using meibography, Kemeriz F. et al. [15] also found a statistically significant higher frequency of MGD and meibomian gland loss among psoriasis patients compared to the control group. Additionally, they found a significant relationship between MGD and psoriasis area severity index (PASI) scores. In a study by Santos da Cruz et al. [16] which included 43 psoriasis patients and 86 control subjects, it was found that patients with psoriasis had a statistically higher rate of blepharitis (16.28%) as compared to control subjects.

Posterior blepharitis remains a clinical diagnostic. It is important to recognize it because it further causes or exacerbates the dry eye syndrome through the tear evaporation mechanism. MGD is present in approximately two-thirds of patients with dry eye disease [17] [18]. This clinical entity is one of the most common ocular manifestations associated with psoriasis. The symptoms of blepharitis are eye itching, burning sensation, and foreign body sensation. The signs of blepharitis are hyperemia, inflammation, and edema at the free margin of the eyelids, with slight scaling [4]. Untreated, chronic blepharitis may lead to ectropion of the lower lacrimal point with epiphora, madarosis, trichiasis, and loss of lid tissue [19,20].

Dry Eye Syndrome

In patients with psoriasis dry eye syndrome (DES) occurs due to an obstructive dysfunction of the excretory ducts of the meibomian glands [4]. The DES may be independent of or may be the result of blepharitis or conjunctivitis [4]. Also, in patients with psoriasis, it was observed that a deficiency of L-arginine may be associated with the decrease of the tear film and DES [21].

DES is maybe the most common ocular manifestation in patients with psoriasis. Zohreh H. et al. [10]. suggest a prevalence rate of dry eye syndrome as high as 18.75%. Singh A. et al. [22] reported an incidence of 27% of dry eye among patients with psoriasis. Another reported incidence was 22% [15].

Beyond the already well-known higher incidence of DES among psoriasis patients, several studies suggested a direct correlation between PASI score and the severity of DES [14]. Aryanian Z. et al. [23] found a significant negative correlation between psoriasis severity based on PASI values and tear meniscus height for both right and left eyes. Also, MGD and MG loss were significantly correlated with PASI [15]. Chimenti M.S. et al. [24] reported even an incidence of DES as high as 75% among patients with psoriatic arthritis, and Allen T.S.R. et al. [25] had an incidence of 60%. Kharolia A. et al. [26] included in their study only patients with PASI scores of more than 10 and reported incidences of 19.1%, 10.2%, and 25% of mild, moderate, and severe dry eye, respectively.

Using a questionnaire to reveal misdiagnosed ocular involvement in psoriasis, Ruggiero A. et al. [3] showed that from a total of 372 patients with psoriasis enrolled in the study, ocular symptoms were detected in 39 patients (10.5%), and 37 patients were referred to ophthalmological examination which confirmed the presence of ocular manifestation in 30 patients. Among these 30 patients, there were 14 cases (46.6%) of dry eye syndrome. Also, given the symptoms of DES including burning, foreign-body sensations, or filmy vision, studies that measured the quality-of-life of psoriasis patients have shown that the impact of moderate to severe dry eye is similar to that of moderate to severe angina [27].

Untreated, this syndrome will complicate itself with keratoconjunctivitis sicca, which could manifest with corneal severe punctate erosions, filamentary keratitis, trichiasis, and even symblepharon or corneal ulceration [27]. Keratoconjunctivitis sicca has been reported at a prevalence rate of 2.7% of psoriatic arthritis patients. [2].

Cornea

Corneal changes in psoriasis are rare, but if present, may be sight-threatening [28]. It has been described peripheral corneal melting, which could lead to perforation [9,29], sterile corneal infiltrates [9], corneal abscess formation, stromal infiltration under Bowman's layer with superficial vascularization and punctuate epithelial keratitis [28,30],

Lee C-Y. et al [31] showed that psoriasis was associated with an increased risk of keratopathy in patients without the preexistent prominent corneal disease, and furthermore, this risk of incident keratopathy increases with exposure to psoriasis.

Starting from the parakeratosis effect of psoriasis, Güneş I.B et al. [32] evaluated the relationship between disease severity and central corneal epithelial thickness. They concluded that central corneal epithelial thickness could be an indicator of psoriasis severity.

Also, corneal hysteresis correlates negatively with disease activity and should be considered when determining intraocular pressure values in glaucoma patients and during corneal evaluation for keratoconus-suspected patients [33].

Furthermore, as shown earlier, it should be remembered that the cornea could be affected by dry eye syndrome which has a higher

prevalence among psoriasis patients [10,15,22].

Internal ocular structures

Uveitis

Uveitis is the inflammation of the uveal tract composed of the iris, ciliary body, and choroid. The main classifications of uveitis are made according to the involved anatomic parts of the uvea (anterior, intermediate, or posterior) or according to etiology (infectious and non-infectious uveitis). Anterior uveitis involves the iris and/or the ciliary body, intermediate uveitis involves the vitreous and posterior uveitis involves the choroid [34].

Uveitis is a complication or a comorbidity of psoriasis [35]. Regularly, psoriasis causes an anterior uveitis [35]. The classic presentation of acute anterior uveitis is the sudden onset of pain, redness, and photophobia that can be associated with decreased vision [35]. Posterior segment involvement can also be present [35]. Uveitis associated with psoriasis tends to be anterior, bilateral, and chronic and tends to appear in older patients than those without psoriasis [36]. The incidence of uveitis tends to be higher in patients suffering from psoriasis and psoriatic arthritis (PsA) or PsA alone [37,38] and could reach 25% [35].

It is not known yet the exact pathological association between psoriasis and uveitis [36]. Possible explanations would be that a breakdown in the barrier between the blood and the aqueous humor could occur as a result of subclinical inflammation in patients with psoriasis, even when the patients do not complain about any ocular symptoms [39,40]. Subsequently, the activated neutrophils in peripheral blood may be responsible for the attacks of anterior uveitis [36]. Also, immune complexes or complement activations mediated by cell surface proteases associated with other chemotactic factors such as leukotriene B4 are found in high levels in the aqueous humor in cases of uveitis in experimental models [36]. Regardless of the mechanism that causes uveitis among psoriasis patients, the close relationship between uveitis and psoriasis has been shown by Chen Y-Y. et al. [41] in a revers manner. They aimed to evaluate whether the risk of subsequent psoriasis and psoriatic arthritis development is increased in patients with uveitis. They found that intermediate uveitis is an indicator of future development of psoriasis.

Activated dendritic cells migrate into draining lymph nodes and secrete tumor necrosis factor (TNF-α), IL-23, and IL-12 [42]. IL-23 and IL-12 will modulate the differentiation and proliferation of Th17 and Th1 cell subsets, respectively [42]. The activation of the adaptive immune response via the distinct T cell subsets drives the maintenance phase of psoriatic inflammation [43].

Regarding these main inflammatory signal cytokines found in the physiopathology of psoriasis, Weinstein et al. [44] studied the relationship between these cytokines and the occurrence of uveitis. In experimental autoimmune uveitis (EAU), both Th17 cells and interleukin (IL) 17 appear to have an important role in driving inflammation. In addition to driving inflammatory cytokine expression, IL-17 signaling also drives the expression of metalloproteases that can cause tissue injury and of chemokines that recruit neutrophils to the site of inflammation [44]. In the adoptive transfer model of EAU, interphotoreceptor retinoid-binding protein (IRBP) specific T cells that express IL-17 are strong effector cells to induce uveitis [45], and treatment with anti-IL-17 antibody is sufficient to block the development of disease [46].

Regarding TNF-α, in the experimental autoimmune uveitis model, neutralization of TNFα suppresses uveitis [47] and the mice with a deficit of TNF receptor 1 are resistant to the development of uveitis [48].

A remarkable feature in the clinical course of psoriasis patients is the presence of HLA-B27. Its importance is due to psoriatic arthritis which belongs to the group of spondyloarthropathies that includes some subgroups for which HLA-B27 is the genetic marker [36]. The relationship between psoriasis, uveitis, and HLA-B27 is not fully established [36]. Instead, there is a clear relationship between positivity for HLA-B27 in psoriasis patients and the severity and incidence of uveitis among them [36]. So, HLA-B27 is considered a risk factor associated with the presence and/or severity of uveitis [36].

The presence of HLA-B27 even has an impact on the cytokines involved in the inflammatory milieu of psoriasis and the intraocular milieu of uveitis. Elevated TNF levels were found in aqueous sampling in patients with HLA-B27 [49,50]. Hernandez Garfella ML. et al. [51] showed that aqueous levels do not appear to reflect the impact of disease activity or treatment with systemic adalimumab. Also, elevated serum IL-23 has been identified as a risk factor for developing uveitis in spondyloarthropathy patients [52]. Regarding data from intraocular studies, the results are mixed. One study did not detect elevated IL-23 in patients with HLA-B27 uveitis analyzing aqueous samples [53]. On the other hand, a proteomic study of vitreous samples from patients with posterior uveitis identified increased IL-23 [54].

Even if uveitis is more common among psoriasis patients with positivity for HLA-B27 and psoriatic arthritis, uveitis can occur in psoriasis without arthritic involvement [55,56]. These findings are supported by other studies [57,58].

Chi C-C et al. [59] concluded that the patients with severe psoriasis without psoriatic arthritis and the patients with mild psoriasis with psoriatic arthritis had an increased risk of incident uveitis. Also, they did not find that patients with mild psoriasis without psoriatic arthritis would have an increased risk for incident uveitis. The higher prevalence of uveitis in patients with psoriatic arthritis patients in comparison with those who suffer only from psoriasis was attributed to the higher frequency of the HLA-B27 seen in patients with PsA [60].

Uveitis is associated with a worse quality of life and greater functional disability [61]. The acute debut of ocular symptoms or signs which could indicate an acute uveitis in a psoriasis patient has to represent a clear indication for an ophthalmological consultation.

Left undiagnosed or untreated, uveitis can lead to posterior synechiae, trabeculitis, pupillary block, macular edema, disc edema [35] or complicated cataract. The risk factors for visual loss are male gender, the presence of posterior synechiae at onset, the use of corticosteroid-sparing therapy or periocular corticosteroid injections, poor control of ocular inflammation, and a chronic course of the disease [62].

Retinal involvement

Regarding retinal involvement of psoriasis, it has been shown that psoriasis patients had a significantly higher risk of developing retinal detachment, retinal vascular occlusion (both arterial and venous), and retinopathy than controls [63].

Psoriasis was suggested itself as a risk factor for retinal vein occlusion regardless of metabolic syndrome [64]. This association relies on two pathologies shared by psoriasis and retinal vein occlusion: metabolic syndrome, considered a risk factor for both psoriasis and retinal vein occlusion [65,66], and homocysteinemia, commonly observed in both psoriasis and retinal vein occlusion patients [67] [68]. Impairment of the retinal vasculature has been demonstrated in several systemic inflammatory diseases, even in the absence of clinical signs of ocular involvement [69,70]. This is why, Castellino N. et al. [71] assessed retinal vascular in psoriasis patients using optical coherence tomography angiography. They showed lower values of vascular density in both superficial and deep capillary plexuses in psoriasis patients compared to controls. Furthermore, another study showed evidence of retinal peripheral vascular leakage in patients with moderate to severe psoriasis [72].

Also, it has been described that birdshot retinopathy can occur in the course of psoriasis [30]. Within posterior uveitis, retina could be affected [35]. Regardless of the cause of posterior uveitis, including psoriasis, retinal involvement could embrace diverse clinical entities such as chorioretinitis, retinochoroiditis, or retinitis [35].

Retinal sensitivity can also be affected in psoriasis [73]. In a study it has been found a positive correlation between disease severity measured through the PASI score and the mean deviations in standard achromatic perimetry and short-wavelength automated perimetry and between disease severity and the corrected pattern standard deviation and pattern standard deviation of short wavelength automated perimetry [73].

As for the electrophysiology function of rods and cones, Shoeibi N. et al. [74] showed that the mean rod response b-wave, standard combined a- and b-waves, single-flash cone response b-wave, and the 30-Hz flicker amplitudes were significantly lower in psoriasis patients than the normal population.

Other ocular manifestations

Because of the inflammatory general status caused by psoriasis, episcleritis can occur in conjunction with psoriasis [2]. Another ocular manifestation described in the literature was a case of posterior scleritis in a patient with psoriasis masquerading as acute angle closure [75]. Also, a case of orbital myositis in a child with psoriasis and juvenile psoriatic arthritis was described by Ajitsaria et al. [76].

Regarding cataract formation, it is not yet known clearly if psoriasis per se causes cataracts, although there are several studies that reported that patients with psoriasis have a higher incidence of cataract development [20,77,78].

The process of cataract formation among patients with psoriasis may be triggered rather by the treatments used in psoriasis [20]. Since this study does not address the side effects of treatments used in psoriasis, we will mention two treatments widely used in psoriasis, which have a well-known link to cataract formation: corticosteroids and psoralen ultraviolet radiation therapy. The systemic use of corticosteroids is associated with the development of posterior subcapsular cataracts [20]. Secondly, psoralen ultraviolet radiation therapy may develop anterior cataracts, because light in the wavelength range of 300-400 nm is absorbed in the lens and may cause photochemical changes in the lens proteins [20].

CONCLUSION

Psoriasis is a complex, multifactorial disease, but most importantly, it is a systemic inflammatory disease, raising its potential to affect ocular structures. Although psoriasis can affect any ocular structure, the exact pathological mechanism of the onset of these conditions is not known. Apart from the postulation of an increased epithelial turnover of the epithelial cells within the meibomian ducts that would cause MGD, the other eye diseases such as uveitis, retinopathies, conjunctivitis, episcleritis still do not present an exact explanation of the increased incidence in psoriasis. On the other hand, although the eye diseases of psoriasis are diverse, more frequent, and more severe than in the general population, they often do not lead to visual disability and have a relatively good prognosis.

Apart from acute episodes of uveitis, retinal detachment, or occlusion of retinal vessels that require urgent ophthalmological care, the rest of the ocular manifestations of psoriasis have an insidious course but, left untreated, lead to a decrease in the quality of life and can become seriously complicated. Therefore, in this context, the ophthalmologist must be included in the multidisciplinary team in all cases of psoriasis patients.

Thus, psoriasis is defined as a disease with multiple associated morbidities, and along with concerns about the relationship with neoplasms [79] or with the adipose-hepatic axis [80], correlations with eye diseases must remain at the top of our concerns.

Conflicts of interest and sources of funding

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Authors' contribution

Conceptualization (MAV, SAA, DOC, LMM); methodology (MP, LMM, DOC); software (ACC); validation (DOC); formal analysis (ACC, LMM, DOC); investigation (MAV, SAA); resources (MAV, SAA, MP); data curation (MP, ACC, LMM, DOC); writing – original draft preparation (MAV, SAA); writing – review and editing of manuscript (MAV, SAA, ACC, DOC); visualization (MP, LMM); supervision (LMM, DOC); project administration (DOC). All authors have read and agreed to the published version of the manuscript.

Ethics approval and consent to participate

Not applicable.

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